

## U.S. Sperm Trend Conclusions

The letter written by Heinze (1) has serious shortcomings. Heinze wrote that

There is not a single study of healthy men from any fertility center or sperm bank that has reported a decline in sperm counts in the United States.

This is not true. A number of such studies exist. Leto and Frensilili (2) documented a decline in sperm counts in potential sperm donors from all over the United States in a longitudinal study.

Heinze stated that

A study by MacLeod and Wang (3) indicates that sperm counts have remained constant in New York since 1938.

That study was dated 1979 and was on men ascertained at a fertility center. Although their sperm counts were stable over the years preceding 1979, it does not necessarily follow that sperm counts of fertile men were stable too.

**William H. James**

Department of Biology  
University College London  
London, United Kingdom  
Fax: 44-0171-383-2048

### REFERENCES AND NOTES

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2. Leto S, Frensilili FJ. Changing parameters of donor sperm. *Fertil Steril* 36:766-770 (1981).
3. MacLeod J, Wang Y. Male fertility potential in terms of semen quality: a review of the past, a study of the present. *Fertil Steril* 31:103-116 (1979).

## U.S. Sperm Trend: Response

I would like to respond to James's comments on my letter "Regional Differences Invalidate U.S. Sperm Trend Conclusions" (1).

Perhaps I should have said that

There is not a single [confirmed] study of healthy men from any fertility center or sperm bank that has reported a decline in sperm counts in the United States.

The study of Leto and Frensilili (2) is contradicted by the four longitudinal studies cited in my letter, which report no decline in sperm counts in five regions of the United States over periods ranging from 10 to 30 years (1). Earlier data on trends in sperm counts were reviewed by MacLeod and Wang (3), who concluded that

enough data have been presented to indicate that there has not been a substantial change in the numerical aspect of semen quality.

Saidi et al. (4), in a recently published review of 29 U.S. studies from the late 1930s to the late 1990s, found "no significant changes in sperm counts during the last 60 years."

MacLeod and Wang (3) reviewed all of the U.S. data available up to that time (1979), including data from fertile men as well as from men evaluated at a fertility center. The earliest data on sperm counts in New York City, published in 1938 (5), were on prenatal couples (i.e., men of known fertility); mean counts ( $137 \times 10^6/\text{mL}$ ) from this study are virtually identical to the mean counts ( $131.5 \times 10^6/\text{mL}$ ) reported in the most recent study from New York City published in 1996 (6), which focused on donors to sperm banks (i.e., men of unknown fertility).

**John Heinze**

John Adams Associates  
Washington, D.C.

E-mail: jheinze@johnadams.com

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## Comments on "Drinking Water Arsenic in Utah: A Cohort Mortality Study"

Lewis et al. (1) compared the mortality of a cohort of members of the Church of Jesus Christ of Latter-day Saints (also known as Mormons) who were exposed to relatively high levels of arsenic through drinking water to the mortality of the general population of Utah. The authors concluded that arsenic exposure may be associated with hypertensive heart disease, nephritis and nephroma, prostate cancer in men, and other heart disease in women. No excess risks were reported for cancers such as those of the skin and bladder, which have been associated with arsenic in other studies (2). We believe that the comparison group used in this study, and the weight given on external rather than internal comparisons, complicates the interpretation of the study results.

Mormons are a selected group that differs from other groups of the general population in many ways, including lifestyle factors such as smoking, which are strong determinants of health. Lewis et al. (1) acknowledged that the study group is known to have about one-half the mortality rates of the general population for diseases such as respiratory and bladder cancers. Given this strong selection bias, it would be unlikely to find any excess risks for these diseases unless this risk associated with arsenic was very high. Similarly, high standard mortality ratios (SMRs) are likely to be caused by other general lifestyle factors, rather than arsenic in drinking water.

When the external comparison group is very different from the index group and information on potential confounders is not available, the best solution is to perform internal comparisons. If conclusions had been based on internal comparisons, neither hypertensive heart disease (SMRs of 2.4, 1.9, and 2.3 for low, medium, and high exposure to arsenic, respectively), nephritis/nephroma (SMRs of 2.0, 2.1, and 0.9, respectively), nor all other heart diseases (SMRs of 2.3, 1.4, and 0.7, respectively) would probably have been associated with arsenic in this study. Among the four causes that Lewis et al. (1) reported to be associated with arsenic, an increasing risk with exposure was only seen for prostate cancer. The authors did mention that internal comparisons are planned. Although such comparisons may be limited by small numbers, any conclusions from this study should await the conduct of such analyses.

**Cristina Villanueva**  
**Manolis Kogevinas**

Respiratory and Environmental  
Health Research Unit  
Institut Municipal d'Investigació Mèdica  
Barcelona, Spain  
E-mail: cvillanueva@imim.es

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1. Lewis DR, Southwick JW, Quellet-Hellstrom R, Rensch J, Calderon RL. Drinking water arsenic in Utah: a cohort mortality study. *Environ Health Perspect* 107:359-365 (1999).
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## "Drinking Water Arsenic in Utah ...": Response

We thank Villanueva and Kogevinas for their letter based on our recent article (1). We agree that to interpret the results of this paper it is important to keep in mind the characteristics of the population used